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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/674,237	07/24/2001	Sean E. Egan	3477-89	4932

20792 7590 07/30/2003

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EXAMINER
DAVIS, MINH TAM B

ART UNIT	PAPER NUMBER
1642	20

DATE MAILED: 07/30/2003

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/674,237	EGAN ET AL.	
	Examiner	Art Unit	
	MINH-TAM DAVIS	1642	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *1* MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *13 November 2002* .

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-58* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☐ Claim(s) _____ is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☒ Claim(s) *1-58* are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

13)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____ .

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a) ☐ The translation of the foreign language provisional application has been received.

15)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449) Paper No(s) _____ .
4) ☒ Interview Summary (PTO-413) Paper No(s). _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: .

DETAILED ACTION

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

The submission of the amendment of 03/29/03 is acknowledged.

After review and reconsideration, the restriction requirement of paper No:16 is vacated, and replaced with the following restriction requirement.

Election/Restrictions

Restriction is required under 35 U.S.C. 121 and 372.

This application contains the following inventions or groups of inventions which are not so linked as to form a single general inventive concept under PCT Rule 13.1.

In accordance with 37 CFR 1.499, applicant is required, in reply to this action, to elect a single invention to which the claims must be restricted.

Group 1. Claims 1-10, 19, 50-53, drawn to an isolated nucleotide sequence encoding a mammalian Ese1 protein, i.e. encoding the murine Ese1 protein of SEQ ID NO:3, or the murine Ese1 nucleotide sequence of SEQ ID NO: 1 and its coding sequence of SEQ ID NO:2, or encoding a human Ese1 protein, a fragment of at least 10 consecutive nucleotides thereof, a complement thereof, a vector comprising said nucleic acid, a host cell comprising said vector, and a method for producing the mammalian Ese1 protein.

Group 2. Claims 1-10, 19, 50-53, drawn to an isolated nucleotide sequence encoding the splice variant murine Ese1L protein of SEQ ID NO:24, or the variant

murine Ese1L nucleotide sequence of SEQ ID NO: 22 and its coding sequence of SEQ ID NO:23, or a splice variant of a human Ese1 protein, a fragment of at least 10 consecutive nucleotides thereof, a complement thereof, a vector comprising said nucleic acid, a host cell comprising said vector, and a method for producing the splice variant mammalian Ese1L protein.

Group 3. Claims 11-17, 39, drawn to a mammalian Ese1 protein, i.e. a murine Ese1 protein of SEQ ID NO:3, a fragment thereof, or a human Ese1 protein.

Group 4. Claims 11-17, 39, drawn to a splice variant murine Ese1L protein of SEQ ID NO: 24, a fragment thereof, or a splice variant of a human Ese1 protein.

Group 5. Claim 18, drawn to an antibody to a mammalian Ese 1, i.e. a murine Ese1, or a human Ese1.

Group 6. Claim 18, drawn to an antibody to a splice variant mammalian Ese 1, i.e. a splice variant of a murine Ese1L, or a human Ese1L.

Group 7. Claims 20-29, 38, 54-57, drawn to an isolated nucleotide sequence encoding a mammalian Ese2 protein, i.e.encoding the murine Ese2 protein of SEQ ID NO:6, or the murine Ese2 nucleotide sequence of SEQ ID NO: 4 and its coding sequence of SEQ ID NO:5, or encoding a human Ese 2 protein, a fragment of at least 10 consecutive nucleotides thereof, a complement thereof, a vector comprising said nucleic acid, a host cell comprising said vector, and a method for producing the mammalian Ese2 protein.

Group 8. Claims 20-21, 23-29, 38, 54-57, drawn to an isolated nucleotide sequence encoding the splice variant murine Ese2L protein of SEQ ID NO:27, or the

murine Ese2L nucleotide sequence of SEQ ID NO: 25 and its coding sequence of SEQ ID NO:26, or encoding a splice variant human Ese 2 protein, a fragment of at least 10 consecutive nucleotides thereof, a complement thereof, a vector comprising said nucleic acid, a host cell comprising said vector, and a method for producing the splice variant mammalian Ese2L protein.

Group 9. Claims 30-36, drawn to a mammalian Ese2 protein, i.e a murine Ese2 protein of SEQ ID NO:6, or a human Ese2 protein.

Group 10. Claims 30-36, drawn to a splice variant mammalian Ese2L protein, i.e.a splice variant murine Ese2L protein of SEQ ID NO:27 or a splice variant human Ese2L protein.

Group 11. Claim 37, drawn to an antibody to a mammalian Ese2, i.e. a murine Ese2, or a human Ese2.

Group 12. Claim 37, drawn to an antibody to a splice variant mammalian Ese2, i.e. a splice variant murine Ese2L, or a splice variant human Ese2L.

Group 13. Claims 40, 42, drawn to a method for screening a candidate compound for treating a disorder characterized by an abnormality in the endocytotic pathway, wherein said pathway involving an interaction between mammalian Ese1, i.e. a murine or a human Ese1, and a binding partner of one of these proteins, comprising screening for the ability of said candidate compound to disrupt said interaction.

Group 14. Claims 40, 42, drawn to a method for screening a candidate compound for treating a disorder characterized by an abnormality in the endocytotic pathway, wherein said pathway involving an interaction between a splice variant of a

murine or a human Ese1 and a binding partner of one of these proteins, comprising screening for the ability of said candidate compound to promote said interaction.

Group 15. Claims 40, 42, drawn to a method for screening a candidate compound for treating a disorder characterized by an abnormality in the endocytotic pathway, wherein said pathway involving an interaction between mammalian Ese2, i.e. a murine or a human Ese2, and a binding partner of one of these proteins, comprising screening for the ability of said candidate compound to disrupt said interaction.

Group 16. Claims 40, 42, drawn to a method for screening a candidate compound for treating a disorder characterized by an abnormality in the endocytotic pathway, wherein said pathway involving an interaction between a splice variant of a murine or a human Ese2 and a binding partner of one of these proteins, comprising screening for the ability of said candidate compound to promote said interaction.

Group 17. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is cancer or abnormal cell division, comprising disrupting or promoting an interaction between a murine or a human Ese1, and a binding partner of one of these proteins.

Group 18. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is cancer or abnormal cell division, comprising disrupting or promoting an interaction between a splice variant of a murine or human Ese1 protein and a binding partner of one of these proteins.

Group 19. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is cancer or abnormal cell division, comprising disrupting or promoting an interaction between a murine or a human Ese2, and a binding partner of one of these proteins.

Group 18. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is cancer or abnormal cell division, comprising disrupting or promoting an interaction between a splice variant of a murine or human Ese2 protein and a binding partner of one of these proteins.

Group 19. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is abnormal cell migration, comprising disrupting or promoting an interaction between a murine or a human Ese1 and a binding partner of one of these proteins.

Group 20. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is abnormal cell migration, comprising disrupting or promoting an interaction between a splice variant of a murine or a human Ese1, and a binding partner of one of these proteins.

Group 21. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said

disorder is abnormal cell migration, comprising disrupting or promoting an interaction between a murine or a human Ese2 and a binding partner of one of these proteins.

Group 22. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is abnormal cell migration, comprising disrupting or promoting an interaction between a splice variant of a murine or a human Ese2, and a binding partner of one of these proteins.

Group 23. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is viral infection, comprising disrupting or promoting an interaction between a murine or a human Ese1, and a binding partner of one of these proteins.

Group 24. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is viral infection, comprising disrupting or promoting an interaction between a splice variant of a murine or a human Ese1, and a binding partner of one of these proteins.

Group 25. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is viral infection, comprising disrupting or promoting an interaction between a murine or a human Ese2, and a binding partner of one of these proteins.

Group 26. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said

disorder is viral infection, comprising disrupting or promoting an interaction between a splice variant of a murine or a human Ese2, and a binding partner of one of these proteins.

Group 27. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is abnormal receptor signaling, comprising disrupting or promoting an interaction between a murine or a human Ese1, and a binding partner of one of these proteins.

Group 28. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is abnormal receptor signaling, comprising disrupting or promoting an interaction between a splice variant of a murine or a human Ese1, and a binding partner of one of these proteins.

Group 29. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is abnormal receptor signaling, comprising disrupting or promoting an interaction between a murine or a human Ese2, and a binding partner of one of these proteins.

Group 28. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is abnormal receptor signaling, comprising disrupting or promoting an

interaction between a splice variant of a murine or a human Ese2, and a binding partner of one of these proteins.

Group 29. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is abnormal tissue development, comprising disrupting or promoting an interaction between a murine or a human Ese1, and a binding partner of one of these proteins.

Group 30. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is abnormal tissue development, comprising disrupting or promoting an interaction between a splice variant of a murine or a human Ese1, and a binding partner of one of these proteins.

Group 31. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is abnormal tissue development, comprising disrupting or promoting an interaction between a murine or a human Ese2, and a binding partner of one of these proteins.

Group 32. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is abnormal tissue development, comprising disrupting or promoting an interaction between a splice variant of a murine or a human Ese2, and a binding partner of one of these proteins.

Group 33. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is abnormal synaptic transmission, comprising disrupting or promoting an interaction between a murine or a human Ese1, and a binding partner of one of these proteins.

Group 34. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is abnormal synaptic transmission, comprising disrupting or promoting an interaction between splice variant of a murine or a human Ese1, and a binding partner of one of these proteins.

Group 35. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is abnormal synaptic transmission, comprising disrupting or promoting an interaction between a murine or a human Ese2, and a binding partner of one of these proteins.

Group 34. Claims 41, 58, drawn to a method for treating or preventing a disorder characterized by an abnormality in the endocytotic pathway, wherein said disorder is abnormal synaptic transmission, comprising disrupting or promoting an interaction between splice variant of a murine or a human Ese2, and a binding partner of one of these proteins.

Groups 35-38. Claim 43, drawn to a method for screening antagonist of a mammalian Ese1, a splice variant of a mammalian Ese1, a mammalian Ese2, or a

splice variant of a mammalian Ese2 protein. A method that screen an antagonist of one of said Ese proteins a single invention.

Groups 39-42. Claims 44-45, drawn to a method for treating a disorder characterized by an undesired level of endocytotic activity of an Ese protein, wherein said disorder is cancer or abnormal cell division, or abnormal proliferation of cells capable of being stimulated to proliferate by a growth factor receptor, comprising administering an antibody that specifically binds to a mammalian Ese1, a splice variant of a mammalian Ese1, a mammalian Ese2, or a splice variant of a mammalian Ese2. A method comprising administering an antibody that specifically binds to one of said Ese proteins constitutes a single invention.

Groups 43-46. Claims 44-45, drawn to a method for treating a disorder characterized by an undesired level of endocytotic activity of an Ese protein, wherein said disorder is cancer or abnormal cell division, or abnormal proliferation of cells capable of being stimulated to proliferate by a growth factor receptor, comprising administering an antisense of a murine Ese1, Ese1L, Ese2, or Ese2L polynucleotide of SEQ ID NO: (1,2), (4,5), (22, 23), or (25, 26). A method comprising administering an antisense of one of said Ese polynucleotides constitutes a single invention.

Groups 47-50. Claim 44, drawn to a method for treating a disorder characterized by an undesired level of endocytotic activity of an Ese protein, wherein said disorder is abnormal cell migration, comprising administering an antibody that specifically binds to a mammalian Ese1, Ese1L, Ese2, or Ese2L protein. A method

comprising administering an antibody that specifically binds to one of said Ese proteins constitutes a single invention.

Groups 51-54. Claim 44, drawn to a method for treating a disorder characterized by an undesired level of endocytotic activity of an Ese protein, wherein said disorder is abnormal cell migration, comprising administering an antisense of a murine Ese1, Ese1L, Ese2, or Ese2L polynucleotide of SEQ ID NO: (1,2), (4,5), (22, 23), or (25, 26). A method comprising administering an antisense of one of said Ese polynucleotides constitutes a single invention.

Groups 55-58. Claim 44, drawn to a method for treating a disorder characterized by an undesired level of endocytotic activity of an Ese protein, wherein said disorder is viral infection, comprising administering an antibody that specifically binds to a mammalian Ese1, Ese1L, Ese2, or Ese2L protein. A method comprising administering an antibody that specifically binds to one of said Ese proteins constitutes a single invention.

Groups 59-62. Claim 44, drawn to a method for treating a disorder characterized by an undesired level of endocytotic activity of an Ese protein, wherein said disorder is viral infection, comprising administering an antisense of a murine Ese1, Ese1L, Ese2, or Ese2L polynucleotide of SEQ ID NO: (1,2), (4,5), (22, 23), or (25, 26). A method comprising administering an antisense of one of said Ese polynucleotides constitutes a single invention.

Groups 63-66. Claim 44, drawn to a method for treating a disorder characterized by an undesired level of endocytotic activity of an Ese protein, wherein

said disorder is abnormal receptor signaling, comprising administering an antibody that specifically binds to a mammalian Ese1, Ese1L, Ese2, or Ese2L protein. A method comprising administering an antibody that specifically binds to one of said Ese proteins constitutes a single invention.

Groups 67-70. Claim 44, drawn to a method for treating a disorder characterized by an undesired level of endocytotic activity of an Ese protein, wherein said disorder is abnormal receptor signaling, comprising administering an antisense of a murine Ese1, Ese1L, Ese2, or Ese2L polynucleotide of SEQ ID NO: (1,2), (4,5), (22, 23), or (25, 26). A method comprising administering an antisense of one of said Ese polynucleotides constitutes a single invention.

Groups 71-74. Claim 44, drawn to a method for treating a disorder characterized by an undesired level of endocytotic activity of an Ese protein, wherein said disorder is abnormal tissue development, comprising administering an antibody that specifically binds to a mammalian Ese1, Ese1L, Ese2, or Ese2L protein. A method comprising administering an antibody that specifically binds to one of said Ese proteins constitutes a single invention.

Groups 75-78. Claim 44, drawn to a method for treating a disorder characterized by an undesired level of endocytotic activity of an Ese protein, wherein said disorder is abnormal tissue development, comprising administering an antisense of a murine Ese1, Ese1L, Ese2, or Ese2L polynucleotide of SEQ ID NO: (1,2), (4,5), (22, 23), or (25, 26). A method comprising administering an antisense of one of said Ese polynucleotides constitutes a single invention.

Groups 79-82. Claim 44, drawn to a method for treating a disorder characterized by an undesired level of endocytotic activity of an Ese protein, wherein said disorder is abnormal synaptic transmission, comprising administering an antagonist or agonist of or an antibody that specifically binds to a mammalian Ese1, Ese1L, Ese2, or Ese2L protein. A method comprising administering an antibody that specifically binds to one of said Ese proteins constitutes a single invention.

Groups 83-86. Claim 44, drawn to a method for treating a disorder characterized by an undesired level of endocytotic activity of an Ese protein, wherein said disorder is abnormal synaptic transmission, comprising administering an antisense of a murine Ese1, Ese1L, Ese2, or Ese2L polynucleotide of SEQ ID NO: (1,2), (4,5), (22, 23), or (25, 26). A method comprising administering an antisense of one of said Ese polynucleotides constitutes a single invention.

Groups 87-90. Claim 47, drawn to a method for promoting endocytosis, comprising administering a mammalian Ese1, Ese1L, Ese2, or Ese2L protein, or an active analogue or mimic thereof. A method comprising administering one of said Ese proteins constitutes a single invention.

Group 91. Claim 48, drawn to a method for blocking clathrin-mediated endocytosis, comprising overexpressing Ese1 protein.

Group 92. Claim 49, drawn to a method for regulating endocytosis, comprising providing an Ese1-Esps 15 complex and further binding said complex to dynamin.

In addition, upon election of any one of groups 1-42, 47-50, 55-58, 63-66, 71-74, 79-82, 87-92, further election of the following patentably distinct species is required:

Murine or mouse Ese.

Upon election of any one of groups 17-34, further election of the following patentably distinct species is required:

Treating or preventing a disorder.

The inventions listed as Groups 1- 91 do not relate to a single general inventive concept under PCT Rule 13.1 because, under PCT Rule 13.2, they lack the same or corresponding special technical features for the following reasons:

A national stage application shall relate to one invention only or to a group of inventions so linked as to form a single general inventive concept. Unity of invention is fulfilled only when there is a technical relationship among the inventions involving one or more of the same or corresponding special technical features which define a contribution over the prior art. If there is no special technical feature, if multiple products, processes of manufacture or uses are claimed, the first invention of the category first mentioned in the claims of the application will be considered as the main invention in the claims, see PCT article 17(3) (a) and 1.476 (c), 37 C.F.R. 1.475(d).

According to PCT Rule 13.2, unity of invention exists only when the shared same or corresponding technical feature is a contribution over the prior art. The inventions listed as groups 1-91 do not relate to a single general inventive concept because they lack the same or corresponding technical feature. The technical feature of group I is a nucleotide sequence encoding a mammalian Ese1 protein. The specification discloses that the mammalian Ese polynucleotide may be in the form of DNA, genomic DNA, cDNA, mRNA, and various "fragments" and "portions" of the gene sequence encoding

Ese protein (p.3, lines 15-17). The claimed mammalian Ese polynucleotide lacks novelty, and does not make a contribution over the prior art, because, SEQ ID NO:38, taught by US 6309820, is 79% similar to the claimed murine Ese1 of SEQ ID NO:3, from amino acid 721 to amino acid 1213, as shown in MPSRCH sequence similarity search (MPSRCH search report, 2002, us-09-674-237a-3.rai, pages 1-2. In other words, a fragment of the gene sequence encoding Ese1 protein, or the claimed mammalian Ese1 polynucleotide is the same as a polynucleotide sequence encoding a fragment of SEQ ID NO:38, taught by US 6309820.

Applicants are required under 35 USC 121 to elect a single disclosed group for prosecution on the merits to which the claims shall be restricted. Applicant is further advised that if Applicant elects a group having species requirement, a response to this requirement must include an identification of the species that is elected consonant with this requirement, and a listing of all claims readable thereon, including any claims subsequently added.

Upon the allowance of a generic claim, applicant will be entitled to consideration of claims to additional species which are written in dependent form or otherwise include all the limitations of an allowed generic claim as provided by 37 CFR 1.141. If claims are added after the election, applicant must indicate which are readable upon the elected species. MPEP 809.02(a).

Should applicant traverse on the ground that the species are not patentably distinct, applicant should submit evidence or identify such evidence now of record showing the species to be obvious variants or clearly admit on the record that this is the

case. In either instance, if the examiner finds one of the inventions unpatentable over the prior art, the evidence or admission may be used in a rejection under 35 USC 103 of the other invention.

Applicants are reminded that upon the cancellation of claims to a non-elected invention, the inventorship must be amended in compliance with 37 C.F.R. 1.48(b) if one or more of the currently named inventors is no longer an inventor of at least one claim remaining in the application. Any amendement of inventorship must be accompanied by a diligently-filed petition under 37 C.F.R. 1.48(b) and by the fee required under 37 C.F.R. 1.17(h).

Any inquiry concerning this communication or earlier communications from the examiner should be directed to MINH-TAM DAVIS whose telephone number is 703-305-2008. The examiner can normally be reached on 9:30AM-4:00PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, ANTHONY CAPUTA can be reached on 703-308-3995. The fax phone numbers for the organization where this application or proceeding is assigned are 703-872-9306 for regular communications and 703-872-9307 for After Final communications.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the receptionist whose telephone number is 703-308-0916.

MINH TAM DAVIS

June 20, 2003

SUSAN UNGAR
PRIMARY EXAMINER